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Correspondence

Serum myoglobin and creatine kinase concentrations in patients with polymyositis or dermatomyositis

SIR, Measurement of serum creatine kinase is an accepted procedure in the follow up of patients with polymyositis or dermatomyositis. This parameter is nearly always raised at some point in the illness, and serves as a useful guide in making decisions regarding therapy. There are preliminary reports on the importance of determination of serum myoglobin levels in such patients, however, suggesting that changes of myoglobin levels may precede alterations of creatine kinase and clinical features.[1] Since the assay for measuring myoglobin is time consuming and expensive and requires the use of radioactive isotopes, it was of interest to perform a prospective study dealing with the possible differences of myoglobin and creatine kinase levels in patients with polymyositis or dermatomyositis and their relevance to the clinical situation.

Eight patients (one male, seven female) entered this study and were followed up for a mean period of 80 weeks (range 23–121 weeks). Five of them had definite polymyositis group I, the others definite dermatomyositis group II, according to the classification of Bohan and Peter.[2] In all patients the diagnosis was confirmed by clinical symptoms, increase of serum skeletal muscle enzymes or proteins (particularly creatine kinase and myoglobin and often aldolase, lactate dehydrogenase, and aspartate and alanine aminotransferases) and characteristic muscle biopsy and electromyographic data.[2] Therapy consisted of prednisone. Five patients were treated according to a definite schedule recently introduced in our centre. Briefly, 1·5 mg/kg body weight per day was administered for a period of three weeks, followed by a daily dose of 1 mg/kg body weight until normal values of muscle enzymes were achieved. Prednisone was then tapered off each month in steps of 5 mg until a daily dose of 15–20 mg was reached. This dose was continued for one year. After that, a tapering schedule was followed in which the dose of prednisone was lowered by 2·5 mg decrements monthly. In three patients the diagnosis of polymyositis or dermatomyositis had already been made, and a non-standardised prednisone treatment was started several months before entering the study. During the study patients were regularly evaluated with respect to their clinical condition, muscle function, and serum levels of creatine kinase and myoglobin.

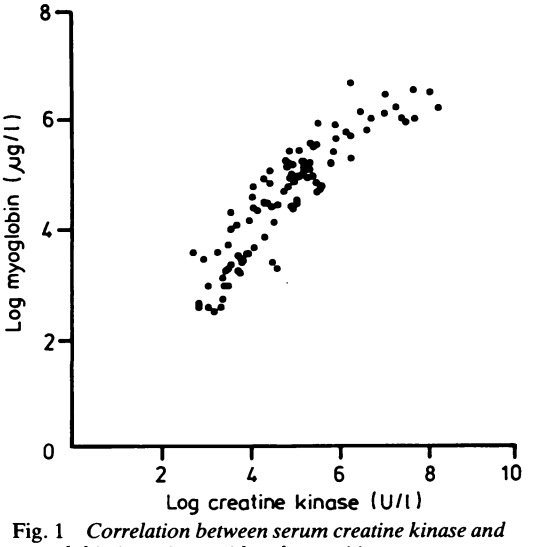

Fig. 1 *Correlation between serum creatine kinase and myoglobin in patients with polymyositis or dermatomyositis.*

One hundred and five sera were screened for creatine kinase and myoglobin. Fig. 1 shows the positive correlation between these parameters. Obviously, this correlation is of significance ($r=0·948$, $p<0·001$). It appeared that by using the myoglobin assay no additional—clinically relevant—information could be obtained since myoglobin and creatine kinase levels correlated equally well with muscle strength and function. In only two cases was an increase of myoglobin concentration found one week before an increase in creatine kinase was observed. Owing to the longitudinal character of this study, however, it could be that this was only incidental and of no importance with respect to the clinical features and therapy. This conflicts with preliminary data from other investigators which indicated that increases in myoglobin levels may precede changes in creatine kinase by several weeks or months and thus could be applied as a useful guide for therapy.[1] Clearly, myoglobin is a more specific indicator of muscle damage than creatine kinase since it is found and synthesised exclusively in skeletal and heart muscle cells[3]; in contrast, creatine kinase derived from non-muscle tissue has been described.[4] The present study, however, does not confirm the advantage of monitoring myoglobin rather than creatine kinase in patients with polymyositis or dermatomyositis. One of the reasons that myoglobin—in spite of its higher muscle specificity—is not a better parameter than creatine kinase in the follow up of such patients is that myoglobin is eliminated very quickly from the blood by renal excretion. Muscle damage due to excessive exercise leads to a rise in myoglobin concentrations within 30 minutes, but the levels return to normal within several hours after exercise.[5] Therefore, myoglobin is probably a very sensitive parameter for monitoring *acute* muscle damage. In patients with polymyositis or dermatomyositis, however, in whom *chronic* muscle damage

occurs, measuring creatine kinase concentration may give a better 'overall' impression of the muscle damage than do myoglobin levels.

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References

1 Nishikai M, Reichlin M. Radioimmunoassay of serum myoglobin in polymyositis and other conditions. *Arthritis Rheum* 1977; **20:** 1514–8.
2 Bohan A, Peter J B. Polymyositis and dermatomyositis. *N Engl J Med* 1975; **292:** 344–7, 403–7.
3 Stone M J, Willerson, J T, Gomez-Sanchez C E, Waterman M. Radioimmunoassay of myglobin in human serum. *J Clin Invest* 1975; **56:** 1334–9.
4 Nevins M A, Saran M, Bright M, Lyon L J. Pitfalls in interpreting serum creatine phosphokinase activity. *JAMA* 1973; **224:** 1382–7.
5 Bär P R, Driessen M, Rikken B, Jennekens F G I. Muscle protein leakage after strenuous exercise: sex and chronological differences. *Neuro Sci Lett* 1985; (suppl 22): 288.

Ankylosing spondylitis and middle ear impairment

SIR, The publication in your journal of a case report by Magaro *et al,* in which a middle ear conductive impairment was observed in association with ankylosing spondylitis (AS),[1] drew our attention to the subject. We studied the auditory function of 94 ears from 48 patients with AS diagnosed according to the New York diagnostic criteria.[2] There were nine women and 39 men, with ages ranging from 15 to 58 years, and an average age of 35 years. In all of them otoscopy, Weber's test, Rinne's test, pure tone audiometry, verbal audiometry, tympanometry, and a stapedial reflex test were performed. In some patients a brain stem evoked response audiometry (BERA) examination was made. Ears in which previous disease could affect the results were rejected (two ears with chronic otitis media). We did not find any middle ear impairment, but sensorineural hearing loss was found more frequently in patients with AS than in the control population.

We think the results presented by Magaro *et al*[1] do not provide sufficient evidence for concluding that the conductive defect was due to involvement of the fibrocartilaginous articulations between malleus and incus or incus and stapes and not to the stapes fixation seen in otosclerotic ears. The case presented by Magaro *et al* could be common otosclerosis in a patient with AS. An exploratory tympanotomy should make it clear which structures are responsible.

Our study suggests that middle ear involvement either does not occur in patients with AS or is very uncommon (less than 1% of ears in patients with AS). On the other hand, the sensorineural component deserves further research, and this is being conducted in our hospital.

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References

1 Magaro M, Ceresia G, Frustaci A. Arthritis of the middle ear in ankylosing spondylitis. *Ann Rheum Dis* 1984; **43:** 658–9.
2 Bennet P H, Wood P H N. Population studies of the rheumatic diseases. *Proceedings of the 3° International Symposium, New York 1966.* Amsterdam: Excerpta Medica, 1968: 456.

Thyroid disorders in systemic lupus erythematosus

SIR, The observations by Goh and Wang in the *Annals* that the prevalence of thyroid disorders is greater in a population of Malaysian patients with systemic lupus erythematosus (SLE) than in the general population[1] are confirmed by our own experience. In a recent survey in Oxford of 64 patients with SLE (61 female, three male) 10 women were found who had also suffered clinical thyroid disease. Seven had had thyrotoxicosis, predating the diagnosis of SLE in six by one to 17 years. All were treated with either carbimazole or Neo-Mercazole, and two required radiolabelled iodine for subsequent flares. Three patients had suffered hypothyroidism, predating the SLE in one, and were treated with thyroxine. Thyroid antibody levels were not available.

The prevalence of overt thyroid disease in our group was 11·5% for thyrotoxicosis and 4·9% for hypothyroidism in women, compared with 1·9 and 1·0% respectively for a British population.[2] There were no clinical features of this subgroup which distinguished them from the total SLE population. Goh and Wang suggest a possible association between the antibodies responsible for the false positive Wassermann reaction and the presence of thyroid disease. None of our thyroid subgroup had a false positive Wassermann reaction, and the frequency of anticardiolipin antibody was no different in these patients from that in the total group. It is possible that the thyroid disorders were related to the presence of thyroid stimulating and inhibiting immunoglobulins which have been demonstrated in patients with SLE.[3]

Two patients showed a third autoimmune disorder; pernicious anaemia in one and Addison's disease in the other. Among the SLE patients without overt thyroid disease were one with insulin dependent diabetes mellitus and one with myasthenia gravis, predating the SLE by 10 and eight years respectively. Clearly there is considerable overlap between the organ specific and non-organ specific autoimmune disorders.

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